Exhibit 13

Northwell Health
Occupational Medicine, Epidemiology and Prevention

August 14, 2017

Moshe Maimon, Esq.
Levy Konigsberg, LLP
800 Third Avenue, 11th Floor
New York, New York 10022

Re: Stephen P. Lanzo

Dear Mr. Maimon:

I am writing to report the results of my evaluation of the materials listed below pertaining to Stephen P. Lanzo. I have reviewed these materials in the context of my pre-existing knowledge, training, and experience in the field of occupational medicine. These materials are of the type I and other specialists in occupational medicine normally rely upon and are sufficient to form a reliable basis for my opinions contained within this report. All of the opinions stated in this report are given within a reasonable degree of medical certainty.

This report and the opinions stated in the report are based on the listed materials and my 25 years of training, education, and experience in the area of asbestos-related occupational medicine. Over the past 25 plus years, I have had the opportunity to evaluate and treat hundreds of patients with asbestos exposure, many of whom have asbestos related diseases.

Qualifications:

I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school in 1988.

I attended the University of Chicago and received a Bachelor of Arts degree with Honors, with a major of History, Philosophy and Social Studies of Science and Medicine. I then continued at the University of Chicago – Pritzker School of Medicine, where I obtained my medical degree in 1988. I was elected to the Alpha Omega Alpha Honor Society, and was also awarded an American Medical Women's Association Award. Following

medical school graduation, I was an intern and resident in Internal Medicine at Yale University – Yale New Haven Hospital from 1988 – 1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991 – 1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times.

Following completion of my residency training in Occupational Medicine, I was awarded a Fellowship in Occupational Medicine from the Foundation for Occupational Health and Research. I continued at Mount Sinai, where I joined the faculty, and continued to evaluate patients with asbestos exposure. I became Vice Chair of the Department of Preventive Medicine in 2001. I was Director of the New York/New Jersey Education and Research Center from 2006 – 2010, and had been Director of the Residency Program in Occupational Medicine from 1998-2006. I was also the Director of the Mount Sinai World Trade Center Medical Monitoring and Treatment Program from 2006 – 2010, although my involvement with the World Trade Center medical programs started in 2001, when I began to evaluate patients with exposure to the World Trade Center disaster, and was initially Medical Core Director of the World Trade Center Worker and Volunteer Medical Screening Program (2002-2004), and Co-Director of the World Trade Center Medical Monitoring and Treatment Program (2004-2006). I have published over fifty articles in the peer-reviewed literature.

In 2010 I left the Mount Sinai School of Medicine to become the Founding Chair of the Department of Population Health at Northwell Health and Hofstra Northwell School of Medicine (formerly known as North Shore University Health System). The Department changed its name in 2014 to Occupational Medicine, Epidemiology and Prevention.

I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning over 25 years. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to patients in the metropolitan New York area. Over the past year alone, I have supervised the examination of or directly examined nearly 500 patients with asbestos exposure, as we have greatly expanded our clinical services. Over the course of the past 25 years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home.

Materials Reviewed:

I have had the opportunity to review the medical records of Mr. Lanzo in order to determine if he suffers from an asbestos related disease. I was provided with the following information:

1. John Muir/Dr. Tsai – Diagnosing pathology report
2. Bay Area Surgical Specialists – Medical Records
3. Baylor St. Luke's Medical Center – Medical Records
4. Contra Costa Oncology – Medical Records
5. COR Cardiovascular Specialists – Medical Records
6. Dr. Henry Kung – Medical Records
7. Honor Health John C. Lincoln Medical Center – Medical Records
8. Houston Methodist Hospital – Medical Records
9. John Muir Medical Center (Walnut Creek) – Medical Records
10. John Muir Medical Center (Concord Campus) – Medical Records
11. John Muir Medical Center (Lafayette) – Medical Records
12. Norwalk Hospital – Medical Records
13. Respiratory Medical Group – Medical Records
14. Scott & White Hospital & Clinic – Medical Records
15. Tahoe Forest Hospital – Medical Records
16. The Lung Institute at Baylor College – Medical Records
17. Vanguard Medical Group – Medical Records
18. Mountainside Family Practice – Medical Records
19. CHI St. Luke's – Pathology Reports
20. Memorial Sloan Kettering Cancer Center – Medical Records
21. Dr. Bakshi (rcvd from client) – Medical Records
22. Dr. Sugarbaker (rcvd from client) – Medical Records
23. John Muir (rcvd from client) – Medical Records
24. John Muir Medical Center – Pathology Report
25. Dr. Andrew Brown Cancer Center - Medical Records
26. Lung Institute at Baylor College of Med (updated) - Medical Records
27. Summit Medical Group Family Medicine - Medical Records
28. Internal Medicine Faculty Practice - Medical Records
29. Advanced Dermatology & Skincare - Medical Records
30. Einstein Medical Center – Medical Records
31. Plaintiff's Answers to Interrogatories Part I – CERTIFIED
32. Depositions of Stephen Lanzo, III – January 16, 2017
33. Depositions of Kendra Lanzo – April 26, 2017
34. Depositions of Michael Lanzo – April 26, 2017
35. Depositions of Margaret McMillan – April 27, 2017
36. Depositions of Robert McMillan - April 27, 2017
37. Social Security Earnings Income Statement for Stephen Lanzo
38. Report of Dr. Zhang, dated July 10, 2017
39. Report of Dr. Ronald Gordon, Mt. Sinai School of Medicine, dated July 20, 2017
40. Report of Dr. Steven Compton, MVA Scientific Consultants, August 1, 2017
41. Report of Dr. William Longo and Dr. Mark Rigler, Materials Analytical Services, LLC, August 2, 2017

42. Asbestos Abatement records from 4 Yale Terrace
43. Rockwool Test Analysis re Asbestos Abatement at 4 Yale Terrace
44. Protected Document, Bates Numbers JNJAZ55_000000797
45. Protected Document, Bates Numbers JNJAZ55_000000840
46. Protected Document, Bates Numbers JNJAZ55_000001014
47. Protected Document, Bates Numbers JNJAZ55_000001032
48. Protected Document, Bates Numbers JNJAZ55_000002190
49. Protected Document, Bates Numbers JNJAZ55_000002203
50. Protected Document, Bates Numbers JNJNL61_1341-1368
51. Protected Document, Bates Numbers JNJNL61_1464-1471
52. Protected Document, Bates Numbers JNJNL61_1480-1484
53. Protected Document, Bates Numbers JNJNL61_1489-1493
54. Protected Document, Bates Numbers JNJNL61_1725-1726
55. Protected Document, Bates Numbers JNJNL61_9799-9813
56. Protected Document, Bates Numbers JNJNL61_10804-10843
57. Protected Document, Bates Numbers JNJAZ55_000001114
58. Protected Document, Bates Numbers JNJAZ55_000002203
59. Protected Document, Bates Numbers JNJAZ55_000005083
60. Protected Document, Bates Numbers JNJAZ55_000011185
61. Protected Document, Bates Numbers JNJNL61_1785
62. Protected Document, Bates Numbers JNJNL61_9483-9485
63. Protected Document, Bates Numbers JNJNL61_9896
64. Protected Document, Bates Numbers JNJNL61_9897
65. Protected Document, Bates Numbers JNJAZ55_000002203
66. Protected Document, Bates Numbers JNJAZ55_000003576
67. Protected Document, Bates Numbers JNJAZ55_000005743
68. Protected Document, Bates Numbers JNJAZ55_000005977
69. Protected Document, Bates Numbers JNJAZ55_000017891
70. McCrone Report on J&J talc dated October 27, 1972
71. Amphibole Content of Cosmetic and Pharma Talcs, Blount, 1991
72. Email re News Reporter Inquiry & Positive Asbestos Finding, February 24, 2004
73. Colorado School of Mines Research Institute's XRD of Vermont talc, July 7, 1971
74. Key to samples in Blount (JNJNL61_000014437)
75. Protected Document, Bates Numbers JNJAZ55_000001587
76. Protected Document, Bates Numbers JNJAZ55_000001892
77. Protected Document, Bates Numbers JNJAZ55_000004643
78. Protected Document, Bates Numbers JNJAZ55_000006060
79. Protected Document, Bates Numbers JNJAZ55_000006088
80. Protected Document, Bates Numbers JNJMX68_000004646
81. Protected Document, Bates Numbers JNJMX68_000004996
82. Protected Document, Bates Numbers JNJMX68_000005032
83. Protected Document, Bates Numbers JNJMX68_000005037
84. Protected Document, Bates Numbers JNJMX68_000006483
85. Document, Bates Numbers ITA-Sabatelli-000548-ITA-Sabatelli-000563

Mr. Lanzo's Medical and Exposure History:

Clinical History: Mr. Stephen Lanzo is a 44 year old man who was treated for a positive tuberculosis skin test (negative chest x-ray) in 2000. He developed chest pain after playing hockey in 2012 and was evaluated in the Emergency Department with a CT scan that showed no pulmonary embolism, pulmonary infiltrates, nodules, effusions or other abnormalities. His cardiac enzymes were negative, and the pain was diagnosed as atypical chest pain. He developed chest pain in April 2014, after exercising and had a cough that worsened the chest pain slightly. He went to see Dr. Kenneth Miller, a cardiologist, on April 1, 2014. His electrocardiogram was normal, and Dr. Miller felt that the chest discomfort was related to minor chest wall trauma from the recent exercise regimen. In December 2014, Mr. Lanzo developed right neck pain radiating down into the shoulder, along with some pins and needles sensations. He was seen in the Emergency Department of John Muir Health in Walnut Creek, CA. An X- Ray obtained of the shoulder and chest was negative. He was treated with anti-inflammatory and pain medication, and then discharged home from the Emergency Department. He was seen by his primary care physician, Dr. Maha Toma, on December 15, 2014, to both establish care, and to evaluate the persistent neck and shoulder pain. Dr. Toma felt that the pain was musculoskeletal and prescribed muscle relaxants. A neck x-ray showed spondylosis in the lower cervical spine with some narrowing and spinal stenosis. Mr. Lanzo returned to Dr. Toma on December 31, 2014. An MRI of the cervical spine showed spondylosis at C6-C7. He was diagnosed with a cervical radiculopathy.

Mr. Lanzo went to the Emergency Department on March 29, 2015, with mid chest pressure that had been building over two days. It did not worsen with activities but was constant. A chest x-ray and electrocardiogram performed were negative. He was subsequently evaluated by a cardiologist who treated him for pericarditis with anti-inflammatory medication. He returned to the Emergency Department on April 6, 2015, with right sided chest pain, numbness, and tingling in his extremities. He had played hockey earlier that night. A chest x-ray showed cardiomegaly with mild central pulmonary vascular congestion. He was discharged from the Emergency Room with a diagnosis of atypical chest pain, and a stress echo was scheduled in three weeks. He returned to the Emergency Department at John Muir Health on April 13, 2015, with symptoms of chain pain associated with a rapid heartbeat and shortness of breath. He also had numbness and burning sensations in his arms. He had frequent night sweats. The chest pain was worse when he was lying down and had not improved with anti-inflammatory medication. There was no acute ischemia on the electrocardiogram, and a chest x-ray showed no acute cardiopulmonary disease. He was placed on a steroid dose pack for possible pericarditis.

Dr. Toma saw Mr. Lanzo on April 20, 2015. He had undergone a complete cardiac evaluation including a stress echo that was unremarkable, with a normal left ventricular ejection fraction, although he continued to have chest pain that he described as band-like; with night sweats that were so severe that he had to change the bed sheets. Dr. Toma ordered a CT scan of the chest, abdomen, and pelvis given the atypical presentation, and referred him to a gastroenterologist, Dr. Henry Kung. Dr. Kung saw Mr. Lanzo, who had also noted some occasional rectal bleeding. Dr. Kung felt that the

rectal bleeding was due to hemorrhoids, but planned to perform an endoscopy and colonoscopy. A CT scan of the chest showed minimal pleural thickening and atelectasis at the right major fissure. There was prior granulomatous disease noted. A 2-millimeter nodule was seen in the left lung. A CT scan of the abdomen and pelvis was unremarkable.

Mr. Lanzo returned to the Emergency Department on June 17, 2015, with recurrent chest pain of two days duration, which Mr. Lanzo described as stabbing in the right anterior chest wall. He was diagnosed with non-cardiac chest pain syndrome. Mr. Lanzo underwent an endoscopy and colonoscopy on June 19, 2015, that showed gastritis and esophagitis. The pathology showed Barrett's esophagitis, and he had internal hemorrhoids and colonic diverticulosis. Dr. Kung placed him on a proton pump inhibitor and high fiber diet. The chest pain persisted, and he returned to the Emergency Department on July 28, 2015. The evaluation was negative, and he was discharged home. M. Lanzo went to see Dr. Wilson Tsai, a gastroenterologist with symptoms of heartburn. Dr. Tsai thought that Mr. Lanzo was having esophageal spasms that were causing the chest pain, and recommended an endoscopy and esophageal manometry. Dr. Tsai performed the endoscopy on August 31, 2015, and found Barrett's esophagus and a widened lower esophageal sphincter consistent with an incompetent lower esophageal sphincter. There was a hiatal hernia with findings consistent with chronic reflux. A chest x-ray on August 31, 2015, showed minor basilar atelectasis with no acute cardiopulmonary process. The pathology from the esophagus showed squamous mucosa with changes consistent with reflux. Esophageal manometry showed peristalsis but only 60% incomplete bolus, with clear evidence of defective lower esophageal sphincter pressures.

Dr. Toma saw Mr. Lanzo on September 25, 2015. He was having recurrent night sweats, two to three times a night. He had undergone an endocrinological evaluation that was normal for catecholamines. He underwent a thyroid ultrasound on October 2, 2015, that showed a nodule in the thyroid isthmus. Dr. Toma saw Mr. Lanzo on October 21, 2015. He had symptoms of dizziness for two weeks. A thyroid biopsy was done on November 6, 2015.

Dr. J. Zaka, a pulmonologist, saw Mr. Lanzo on November 17, 2015, for evaluation of his abnormal CT scan. At the time of the visit, Mr. Lanzo noted an occasional cough and on/off chills. He did have some chest wall discomfort associated with positions and movement. Dr. Zaka planned a repeat CT scan in one year to monitor the nodule. Mr. Lanzo developed chest pain, discomfort, and shortness of breath. He went to the Tahoe Forest Hospital District Emergency Department on November 28, 2015. His electrocardiograms were negative.

Mr. Lanzo was seen at John Muir Health by Dr. Herbert Ure on December 22, 2015. He had sharp right chest pain that was present under his right arm and had pain taking a deep breath. This pain had been present for two weeks. He also noted a white patch on his lower lip. Dr. Ure noted that pain was clearly in the chest wall and referred him to a dentist and dermatologist for the lip lesion. A chest and rib x-ray performed on

December 22, 2015, was negative for rib injury or acute cardiopulmonary disease. Dr. Toma saw Mr. Lanza on December 28, 2015. He still had chest pain that was worse with sneezing and movement. He had tenderness at the right costochondral junction at the 3rd and 4th rib. He was treated with anti-inflammatory medication. Mr. Lanzo developed migraine headaches and saw Dr. Toma on February 10, 2016. She ordered a CT scan of the head and sinuses. The head CT was normal.

Dr. Zaka saw Mr. Lanzo on February 22, 2106. Mr. Lanzo complained of a cough for two months and persistent night sweats. Dr. Zaka ordered a repeat chest CT scan. The CT scan performed on February 25, 2016, showed increased pleural thickening or non-calcified pleura plaque along the right major fissure and anterior right hemithorax along the right upper lobe. Benign or malignant pleural disease should be considered, and PET/CT or tissue sampling was recommended by the radiologist. Dr. Zaka saw Mr. Lanzo on March 10, 2016. He ordered a PET scan based on the CT scan findings. Dr. Tsai saw Mr. Lanzo on March 11, 2016, and recommended an endoscopy with laparoscopic nissen fundoplication and repair of the diaphragm. A PET/CT scan was done on March 17, 2016, and showed unilateral hypermetabolic pleural (fissural and non-fissural) soft tissue abnormalities, suspicious for malignancy involving the pleura. There were non-specific tiny parenchymal lung nodules, one being calcified that were stable. The radiologist recommended a cardiothoracic surgical consultation for tissue sampling of the pleural thickening. Mr. Lanzo returned to Dr. Zaka on March 22, 2016. He referred Mr. Lanzo to Dr. Tsai for a tissue biopsy based on the PET scan findings. Dr. Tsai saw Mr. Lanzo on March 23, 2016, and recommended a flexible bronchoscopy, right video thoracoscopy (VATS), and excisional biopsy of the pleural mass.

Mr. Lanzo underwent a bronchoscopy and VATS with an excisional biopsy on March 29, 2016. At the time of surgery, Dr. Tsai noted that there were pleural masses with extension of tumor into the lung. He excised the pleura lesion and performed a wedge biopsy of the right lung. The frozen specimens showed malignant cells. The pathology showed malignant mesothelioma, epithelioid cell type, with invasion into the skeletal muscle. The lung mass showed metastatic malignant mesothelioma, epithelioid cell type, grossly present at the surgical resection margin. Mr. Lanzo was discharged on March 30th, 2016. Mr. Lanzo was referred to Dr. Michael Sherman, a medical oncologist, who saw him on April 1, 2016. He was still having intermittent palpitations with chest pain, occasional dry cough, and chest pain on the right side. Dr. Sherman recommended chemotherapy with Alimta, Cisplatin, and Avastin. Mr. Lanzo received his first cycle of chemotherapy on April 8, 2016.

Mr. Lanzo went to see Dr. Sukhmani Padda at Stanford Center on April 11, 2016. He had a tough time after receiving the chemotherapy and had a cough and mild chest pain. Dr. Padda noted that Mr. Lanzo's tumor appeared noncontiguous and the skeletal muscle and lung invasion made the case less straightforward. He agreed with chemotherapy followed by surgical evaluation if there was a response. Dr. Tsai inserted a venous access port on April 12, 2016.

Dr. Sugarbaker saw Mr. Lanzo on April 25, 2016. He noted that his disease volume was low and that he appeared to be a good surgical candidate. Dr. Sugarbaker recommended that Mr. Lanzo stop his chemotherapy and complete his surgical staging with a mediastinoscopy and laparoscopy. Pulmonary function tests were normal, and he had no oxygen desaturation with a six minute walk. A cardiac echo showed normal heart function with an estimated left ventricular ejection fraction of 65%. An MRI showed small patchy enhancing areas of minimal pleural thickening, mainly in the right medial lower hemithorax. The tumor abutted the spinous process of T10, but did not invade into the foramina. There was an abnormal MRI signal intensity in the lateral aspect of the right fourth rib in the region of the previously noted subpleural nodule that could represent tumor involvement. There was a faint 2-millimeter focus adjacent to the posterior right atrium. Dr. Sugarbaker felt he was a good surgical candidate. Because his white blood cell count was low, he planned to wait for one to two weeks. A quantitative ventilation perfusion scan showed 45% perfusion to the left lung and 55% to the right lung, and no evidence of embolic disease or parenchymal lung disease.

Dr. Theirry Jahan at UCSF saw Mr. Lanzo on April 29, 2016. The pathology was reviewed at UCSF Medical Center, and the diagnosis of malignant mesothelioma was confirmed. Dr. Jahan recommended continuing with chemotherapy for a total of three to four cycles prior to surgical intervention (assuming no disease progression).

On May 5th, 2016, Mr. Lanzo underwent a mediastinoscopy and laparoscopy. There was metastatic spread to the level VII lymph node. There was no tumor noted on peritoneal biopsies or peritoneal washings. Given the positive lymph node, Dr. Sugarbaker recommended that he resume chemotherapy. Mr. Lanzo had right shoulder, rib, and flank pain after returning from Baylor Medical College on May 6, 2016. He was evaluated in the Emergency Department at John Muir Health that evening, and a CT angiogram showed no evidence of a pulmonary embolism. There was a 5% apical pneumothorax. A follow-up chest x-ray on May 10th showed an improvement in the pneumothorax.

Dr. Sherman saw Mr. Lanzo on May 13, 2016, and resumed chemotherapy, given the positive mediastinal lymph nodes. Dr. Zaka saw Mr. Lanzo on May 17, 2016. Dr. Tsai saw Mr. Lanzo on May 18th. He felt a small bulge when he coughed, which Dr. Tsai thought might be scar tissue. Dr. Sherman saw him on May 20th, 2016. Mr. Lanzo had delayed nausea, for which Dr. Sherman prescribed a Nomotex device, and he was encouraged to use Ativan at night. Mr. Lanzo received chemotherapy on June 6, 2016. A chest x-ray on June 9, 2016, showed a right apical pneumothorax that had increased in size compared with May 27th. Dr. Tsai saw Mr. Lanzo on June 10th; he recommended continued observation of the air bulge at the incision site.

Dr. Mark Berry, a thoracic surgeon at Stanford, saw Mr. Lanzo on June 15th. He had intermittent nausea and vomiting, as well as an ongoing persistent dry cough, sore throat, and fatigue. He had slight swelling around the old chest tube site when he coughed, around the size of a golf ball. Dr. Berry noted that Mr. Lanzo did not have disease progression and felt it would be feasible for surgery given his young age and

being physical fit. Dr. Berry recommended that Mr. Lanzo pursue surgery with intrapleural chemotherapy. Mr. Lanzo developed abdominal, and lower chest pain then went to the Emergency Department on June 22, 2016. The pain was worse with breathing. A CT-angiogram showed no evidence of pulmonary artery embolic filling defect in the main or proximal segment pulmonary arteries. There were a few patchy ground glass opacities in the posterior aspect of the right upper lobe, which was non-specific and might be related to infectious or inflammatory etiology. Mr. Lanzo's electrocardiogram showed findings that were consistent with early pericarditis. He was started on Motrin.

Dr. Jahan saw Mr. Lanzo on June 28th. He noted that Mr. Lanzo had difficulty tolerating the chemotherapy with acute pain in his throat and persistent pain upon swallowing. He had mild hemoptysis with Avastin. Mr. Lanzo was also having regular night sweats, intermittent discomfort, and pain in his right chest. Surgery was planned for late July after a repeat PET scan to ensure there was no spread to the chest cavity. Dr. Jahan recommended evaluation by ENT but felt it was likely irritation from intubation and exacerbation by the chemotherapy. A PET/CT scan on July 8, 2016, showed a near complete metabolic and anatomic response to systemic chemotherapy with regards to the right hemithorax. There was minimal residual abnormality in the levels of hypermetabolism. There was some uptake in the supraglottic region. Dr. Sherman saw him on July 14th. He recommended a follow-up with an ENT.

Mr. Lanzo was admitted to St. Luke's Medical Center (Baylor) on July 26, 2016. He had a weak voice since chemotherapy; he noted that the hematemesis presented after chemotherapy had improved. Pulmonary function tests showed normal function. On July 27, 2016, Mr. Lanzo underwent a right pleurectomy and decortication. At the time of surgery, Dr. Sugarbaker found a relatively low burden of disease in the chest with no disease seen on the visceral pleura. He had a parietal pleurectomy, and the visceral pleurectomy was done along the fissure between the upper and lower lobe. There was argon beam ablation of the diaphragmatic surface and of suspicious foci in the lung. A complete ablation of the chest wall was done with the argon beam, and a chemical scrub and heated intra-operative chemotherapy was instilled with Cisplatin. Four chest tubes were placed. Mr. Lanzo received three units of blood and two units of fresh frozen plasma. The pathology showed extensive malignant mesothelioma, epithelioid type, in the visceral and parietal pleura and focally in the connective tissue of the parietal pleura. There was spread to the dermal region of the previous inferior right chest tube site. Mr. Lanzo developed an acute thrombus in the right upper extremity (superficial cephalic vein). There were no deep venous thromboses in the lower extremities or deep venous system. Mr. Lanzo developed severe nausea that was not responsive to anti-emetics, and he required aggressive volume resuscitation on the first postoperative day, followed by diuretics on the second postoperative day. He had a nasogastric tube that was removed on the third postoperative day, and his nausea improved significantly. He was discharged home on August 5, 2016.

Pulmonary function tests on August 9, 2016, showed normal function, but a decrease from postoperative values (forced expiratory volume in the first second

decreased from 4.75 liters, 106% of predicted, to 3.66 liters (89% of predicted). A chest x-ray on August 11th, showed post-surgical changes with a trace right pleural effusion versus pleural thickening. Dr. Sugarbaker saw Mr. Lanzo on August 11, 2016. He had fatigue with activity but was otherwise doing well. He had a second postoperative visit on August 16th. He was active and walking around, but remained fatigued. Mr. Lanzo developed a fever and had a wound infection on August 28th. The wound was opened, and he was placed on Cephalexin, an antibiotic.

A chest x-ray on September 6th showed unchanged right lung pleural thickening versus a small right pleural effusion. Dr. Sugarbaker saw Mr. Lanzo on September 6, 2016. He was on antibiotics for a wound infection with a previous port site that was open and clear, and a seroma of the skin near the port location. Dr. Sugarbaker recommended an additional three cycles of chemotherapy after he had recovered from surgery. Mr. Lanzo was to return in three to four weeks for a repeat CT scan and to see if it were necessary to drain the seroma and then resume chemotherapy. Mr. Lanzo developed fever, chills, nausea, vomiting, and went to the Emergency Department on September 17, 2016. He felt as if fluid was collecting in the right axillary area, and he had some abdominal discomfort. His chest x-ray sowed a blunted right costophrenic angle consistent with a pleural effusion and/or elevated right hemidiaphragm. His wound appeared to be healing well. He was discharged home. Dr. Sherman saw Mr. Lanzo on September 20, 2016. He prescribed Tessalon perles for an extensive cough. Dr. Sherman planned to see him back after he was cleared by the surgeon to resume the chemotherapy.

A CT scan on September 26, 2016, showed an interval right pleurectomy for resection of a malignant pleural mesothelioma. There was no recurrent disease in the chest. There were post-thoracotomy changes with free air in the right chest. Dr. Sugarbaker saw Mr. Lanzo on September 27th. His pulmonary function had improved to 97% of predicted. He recommended that Mr. Lanzo resume chemotherapy, and also attend physical therapy for his right hand and shoulder, given the feeling of tightness in the area. Dr. Sherman saw Mr. Lanzo on September 30, 2016. Mr. Lanzo felt as if there were fluid in his chest when he coughed. He had 5/10 pain. He received chemotherapy on September 30, 2016, and then returned to Dr. Sherman's office on October 5th for hydration. Mr. Lanzo had severe fatigue, nausea, and vomiting with the chemotherapy and Dr. Sherman tried Zyprexa since the other medications did not work.

A chest x-ray on October 6, 2016, showed a recurrence of the right pneumothorax with a 3 centimeter separation between the visceral pleura and the dome of the diaphragm, with increased herniation of intrathoracic contents posterolaterally in relation to the thoracotomy site with increased size of a subcutaneous air-filled bulging deforming the overlying skin surface. There was no left pneumothorax. There was also an interval increase in the amount of deep soft tissue air in the right chest wall located external to the rib cage. An ultrasound of the chest showed no solid mass or fluid collection at the site of palpation in the posterior right thorax. There was an echogenic shadowing structure that could be soft tissue callus, protuberant bone, or herniated lung tissue at the surgical site. He developed increasing swelling near the thoracotomy site on October 7th, along with increasing tightness and some mild increase in the shortness of breath. A CT scan showed

a loculated right basal pneumothorax estimated at 30% with air herniating through the right lateral chest wall and causing subcutaneous emphysema. There was some irregular visceral pleural thickening with adhesion and tenting of the right middle lobe pleura. Areas of more focal pleural thickening were present along the chest wall posteriorly, laterally and along the right middle lobe visceral pleura. There was slightly increased pleural thickening along the right oblique fissure. There was minimal pleural fluid or thickening in the right posterior costophrenic sulcus. There were some compressive changes and subsegmental atelectasis of the right lung without acute infiltrates. Mr. Lanzo was discharged from the Emergency Department and was told no intervention was required for the pneumothorax, which was expected to diminish over time. He returned to the Emergency Department the following day with discomfort when he coughed, noting that the soft tissue swelling on his low back expanded and he felt that there was communication between the soft tissue and his pleural space. The pain was 6/10 in intensity. A chest x-ray showed a slightly decreased right basilar pneumothorax and decreased air in the right chest wall soft tissues. There was right basilar atelectasis.

Dr. Berry saw Mr. Lanzo on October 12, 2016. He had some mild shortness of breath associated with the subcutaneous emphysema and still had night sweats. A chest x-ray showed a moderate sized mostly basilar right pneumothorax. Dr. Berry felt it was somewhat smaller than the October 7th CT scan. Dr. Berry advised Mr. Lanzo not to fly until the pneumothorax had resolved, and planned a repeat chest x-ray in one week. A chest x-ray on October 14th, showed a stable right basilar pneumothorax occupying approximately 5-10% of the total right lung volume. There was no significant interval change. Dr. Tsai saw Mr. Lanzo on October 14, 2016, who noted stale findings and recommended continued observation. Mr. Lanzo returned to Dr. Sherman on October 21, 2016. Mr. Lanzo was neutropenic, and was prescribed Neulasta, and chemotherapy with Cisplatin and Pemetrexed was delayed one week. A chest x-ray on October 27, 2016, showed a decrease in the right subpleural pneumothorax. Mr. Lanzo was having severe headaches, with vomiting and dizziness. A brain MRI was negative, and he was advised to use over the counter analgesics. He received his fifth cycle of chemotherapy on October 28, 2016.

A chest x-ray on November 1, 2016, showed resolution of the previously noted right-sided pneumothorax, and a decrease in the right sided subcutaneous air collection along the right chest wall. Mr. Lanzo was seen in the Emergency Department on November 1, 2016, with nausea, weakness, fatigue, and vomiting. He had numbness in his left hand, and it felt cold. An ultrasound of the left arm showed no evidence of a deep venous thrombosis. He was given one liter of fluid. Dr. Sherman evaluated Mr. Lanzo on November 4, 2016. Mr. Lanzo had lost 10 pounds over the prior seven days and had not been eating. Dr. Sherman sent him to the Emergency Department for admission and discussed holding the Cisplatin off for future cycles due to the severity of his symptoms. He was hospitalized on November 4th, 2016 at John Muir Health for intractable nausea and vomiting. He was treated with intravenous fluids and antiemetics, which improved after two days. A CT scan of the abdomen and pelvis showed no evidence of ileus or obstruction. He received potassium replacement for a low level.

Mr. Lanzo developed chest pain on November 12, 2016, and went to the Emergency Department at Baylor Scott and White Medical Center. Mr. Lanzo described the pain as dull, in the lower sternum. A chest CT angiogram showed multiple subsegmental pulmonary emboli. He received a heparin bolus and infusion. A lower extremity Doppler was negative for deep venous thrombosis. He was discharged home on November 13th on Lovenox. A chest x-ray on November 15, 2016, showed no significant interval change. Dr. Sugarbaker saw Mr. Lanzo on November 15th. Mr. Lanzo had flown to New York and Dallas the prior week. He was now on Lovenox for pulmonary emboli. A PET/CT scan showed postoperative changes consistent with a right thoracotomy and pleurectomy. There was no pleural thickening or increased FDG activity suspicious for residual and recurrence. There was increased FDG activity in the esophagus that was likely esophagitis.

Mr. Lanzo returned home from Dallas to California on November 15th, but developed sternal pain and got off the plane in Phoenix, concerned that his condition was worsening. A CT angiogram was done at Honor Health that showed no pulmonary emboli. There was a linear radiopaque density in the lower portion of the right major fissure with associated finding of multiple old fracture defects in the right lateral chest wall and mild thickening in the parietal pleura. He was cleared to fly home to California. Dr. Sherman saw Mr. Lanzo on November 18th. He received his sixth cycle of combination chemotherapy but received a dose reduction in the Cisplatin due to side effects. He was to receive hydration the day after chemotherapy and the following week. Dr. Sherman felt that Mr. Lanzo should receive lifelong anti-coagulation and switched him to Xarelto. He was admitted overnight to the hospital on November 23, 2016, for chest pain, nausea, vomiting, and received intravenous hydration and antiemetics. A CT-angiogram showed no pulmonary embolus or aortic dissection. He had postsurgical changes in the right chest and no pneumothorax. There were small tree-in-bud nodules in the right middle lobe. He was discharged home the next day.

Dr. Zaka saw Mr. Lanzo on December 13, 2016. He was advised to continue his medications. Mr. Lanzo went to St. Barnabas Medical Center on December 20, 2016, with shortness of breath. A CT scan showed no pulmonary emboli, but there was a small subcentimeter right major fissure nodule. An outpatient stress test was normal. Dr. Kristin Fless, a pulmonologist in New Jersey, saw Mr. Lanzo on December 28, 2016. Mr. Lanzo went to establish care with a local physician. He noted some blood in mucus in the morning and GERD/mucus sensations when he was lying flat at night. Dr. Fless thought the sputum with streaks of blood might be related to the upper airway. She planned to follow him in conjunction with Dr. Sugarbaker. He had a normal cardiac stress test on January 2, 2017, with normal heart function and no evidence of ischemia on the SPECT portion. A CTA on January 2, 2017 showed no evidence of an acute pulmonary embolism. Mr. Lanzo was evaluated by Dr. Andrew Brown, a medical oncologist in New Jersey on January 9, 2017. Mr. Lanzo had an endoscopy on January 17, 2017 that showed mild chronic gastritis and no evidence of Barrett's esophagus.

Dr. Lanzo was evaluated by Dr. Marjorie Zauderer at Memorial Sloan Kettering on January 23, 2017. His pathology had been reviewed prior to the appointment, where the diagnosis of stage III, T3pN2M0 epithelioid mesothelioma was noted. He had intermittent night sweats that had improved since chemotherapy. He had intermittent pain in his chest at the site of the surgery, and also had sharp neuropathic pain in his fingers and toes. He had mild shortness of breath. Dr. Zauderer noted that he had received full treatment, and did not advocate for maintenance chemotherapy given that he had undergone resection. She recommended continuing the Xarelto and planned to monitor him. She also discussed possible radiation and referred him to Dr. Zimner. Dr. Sugarbaker saw him on January 27th. He had some chest tightness and midline discomfort with exertion that was limiting his exercise. A chest CT scan on January 27th showed a stable right pleurectomy with no disease recurrence. There was no pleural effusion or pneumothorax. Mr. Lanzo was seen at Baylor for follow-up on January 30, 2017. He had some peripheral neuropathy in his finger tips and toes and his night sweats were improving. He had some chest tightness and midline discomfort with exertion that was limiting exercise. A venous Doppler study of the lower extremity on February 2, 2017 showed no deep venous thrombosis.

Mr. Lanzo returned to Baylor for a follow-up evaluation on April 11, 2017. He was having headaches and dizziness. He still had occasional chest tightness and peripheral neuropathy. He also had hair loss on his legs, buttocks and scalp. A chest CT scan showed no definite evidence of recurrent disease. There was a 1.1 centimeter fluid density nodule adjacent to the mid-esophagus that was more conspicuous than before but did not appear to be concerning. A brain MRI showed no intracranial abnormalities or metastases. Mr. Lanzo had a chest x-ray and CT angiogram done on June 30, 2017. There was no evidence of a pulmonary artery aneurysm or embolism. There was a 7-millimeter focal area of opacity in the left upper lobe that was in the subpleural location. Mild increased interstitial markings were noted. Postoperative changes were seen in the right chest without change, and there was no evidence for a recurrent mass.

I had the opportunity to evaluate Mr. Lanzo on July 6, 2017. He noted that he still has episodes of labored breathing with chest pain episodes, including one the prior week. He finds that he has to catch his breath. He is fatigued at night and exhausted by 7:30-8 pm. He had a morning cough but no pleuritic pain. He had pain lying on his right side and had to lie on his left side only. He had severe neuropathy in his fingers and toes until March 2017 that extended to his hands and arms. He described "odd" chest pains in his right chest. Mr. Lanzo still had some swelling in his ankles and calves. He also had night sweats that were still present, but in general not as severe. Mr. Lanzo described his illness as taking a psychological toll on himself and his family. He states that he "doesn't feel himself" and has pain just walking around that he lives with day to day.

Mr. Lanzo was evaluated at the ED at Albert Einstein Medical Center in Germantown, PA on July 26, 2017. Mr. Lanzo had left sided chest discomfort, with a squeezing sensation in his biceps and cramping legs. His legs also felt lethargic. A chest

x-ray showed post-surgical changes with no active disease. His laboratory work was normal, and he was advised to follow-up with his primary care physician.

Past Medical History: Mr. Lanzo has no significant medical history; he was diagnosed with Barrett's esophagus/esophagitis. He is on Omeprazole and Xarelto.

Cigarette Smoking History: Mr. Lanzo was a non smoker.

Occupational and Environmental History: Mr. Lanzo's first job was working in the Montclair Library stacking books while he was in high school. He also worked as a cashier and stock boy at the supermarket as well as the computer lab while in college. His father and stepfather were accountants, and his mother was a teacher. He graduated from college in 1994 and had worked for various firms in the financial industry since 1994. He is currently working for Bank of America/US Trust.

Mr. Lanzo noted that home renovations were done to refinish the attic in the family home in 1984. His stepfather did the work, using sheetrock and joint compound. Insulation was used and tested; it was rock wool with no asbestos detected. Asbestos abatement was done in his childhood home in 2002 (Mr. Lanzo was not present and had not regularly lived in the home for over a decade), with removal of 60 linear feet of exposed asbestos containing pipe insulation.

Mr. Lanzo was exposed to Johnson and Johnson talcum powder from an early age. His mother applied the talcum powder to him daily after his bath until he was able to apply it himself at around age 6. He recalled that he used to play with the powder with his brothers and that his mother also used the powder, which he described as "dousing her body with it." Mr. Lanzo's chore was cleaning the bathroom, where the family applied the talcum powder every week. Mr. Lanzo recalled using the talcum powder in the bathroom or his room, and that there would be powder on the floor. He had to vacuum up the powder from his room on a weekly basis. He applied the talcum powder to his torso, groin, legs and back, often twice a day after showering. His poured the powder into his hand and applied it, or poured it directly from the container onto his chest and rubbed the powder. He recalled getting mouthfuls of powder during the application. Mr. Lanzo played ice hockey and used the powder in his gloves, shoulder protector, skates, and shoes. He had a daily practice or every other day practice in middle school and high school and then three times a week while in college. He used powder after showering when he completed his ice hockey practices/games. He also used Shower to Shower powder while in high school and college in addition to the Johnson and Johnson baby powder. He went through a contained of talcum powder every two weeks. His wife noted that Mr. Lanzo used so much powder on his chest that his chest hair was white. Mr. Lanzo's brother also used talcum powder after his bath and shower in the bathroom they shared.

Physical Examination: Mr. Lanzo is a well-developed, well-nourished man appearing his stated age. His blood pressure was 114/75, his pulse was 65 beats per minute, and his respiratory rate was 15 per minute. His oxygen saturation was 97%. Examination of his

head and neck, heart, abdomen, and extremities were normal. He had several scars on his chest from the four chest tubes, the thoracotomy, the thoracoscopy, laparoscopy, and mediastinoscopy. He had slightly decreased breath sounds on the right side and noted pain in the right upper chest with deep breathing.

Conclusion: Mr. Lanzo is an unfortunate 44 year old man with malignant mesothelioma of the right chest as a result of his exposure to asbestos from contaminated talcum powder. He has undergone state of the art treatment, including surgical resection and chemotherapy. His tumor has spread to his lymph nodes.

Based on the information available, it is my opinion, to a reasonable degree of medical certainty that Mr. Lanzo's exposure to asbestos-containing talcum powder led to the development of his mesothelioma. He began using Johnson and Johnson talcum powder in 1972 and continued using it until 2015. He also used Shower to Shower talcum powders in the 1980s and 1990s.

The methodology and basis for my opinions follows standard methods of the medical and scientific community. Asbestos is the most well known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both worked directly with asbestos products and for those who worked adjacent to or in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

Asbestos and Malignant Mesothelioma General Opinions: Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard

medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may-but doesn't always-include identifying the patient's occupation.

A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not been supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasis what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease: Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to *any* asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize

today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. The ambient air concentration or "background level" has been reported to range from 0.0005 f/cc in urban areas, to 0.00005 f/cc in rural regions. These levels are thousands of times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art:
In 1898 Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

In 1930 Merewether and Price, in their *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*, noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studies asbestos workers in the textile mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure and was published by Wedler in Germany. Also in 1943, Hueper from the United States Public Health Service stated that he believed

asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In 1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with asbestos exposure. In fact, by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

The Development of Diseases: When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis: The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5th complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts, myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.*,19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues*., *19* Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth

and matrix production that we recognize as asbestosis. Like *all* of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis: This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer: These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a

malignant tumor. (See Frost G, Darton A, Harding AH. *The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005)* Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma: This cancer occurs when a mesothelial cell of the pleural or peritoneal surfaces develops a sufficient number of genetic errors in a set of genes that control cell growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study,* 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. *Malignant pleural mesothelioma: an epidemiological perspective,* 1 Annals Cardiothoracic Surgery 491 (2012)).

Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer.

All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, *Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma.*, 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. *Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.*, 208 Int'l. J. Hygiene Env. Health 201 (2005)). Some have suggested that geological nomenclature – calling the anthophyllite and tremolite in the talc either "non-asbestiform" or "cleavage fragments" – has biological significance. This notion has been rejected by the EPA, US Centers for Disease Control and Prevention, Agency for Toxic Substances and Disease Registry, and American Thoracic Society, and is not a distinction that is considered medically important. In fact, mesotheliomas have been documented among New York State miners and millers of talc containing approximately 50% "non-asbestiform" anthophyllite and tremolite. Asbestos related diseases have also been found at the Vermont talc mines and mills. The absence of documented cases of mesothelioma among one cohort of miners and millers of talc containing less than 1% the tremolite and anthophyllite (such as the Italian studies of talc

miners and millers) is most likely due to an inadequate sample size. (US EPA Region 9 Response to the 2005 National Stone, Sand and Gravel Association Report, April 20, 2006; RT Vanderbilt Co., MSDS, May 1, 1975; Roggli, et.al. *Tremolite and Mesothelioma.*, Ann Occ Hyg 46(5):447-453 (2002); Lamm, *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers;* Epidemiology-Fibers, 1576-1581 (1988)).

Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., *Developments in Asbestos Cancer Risk Assessment.* Am J. of Indus. Med. (2009)).

Moreover, there is ample evidence to support the conclusion that exposure to the asbestos fibers typically used in brake linings-chrysotile fibers-can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sizes can cause mesothelioma is also reflected in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review*, 44 Am J. Indus. Med. 291 (2003); D. Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies.* 44 Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. *Fiber Types, Asbestos Potency, and Environmental Causation.* 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look*, 52 Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos.* 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., *The Hazards of Chrysotile Asbestos, a Critical Review.* 37 Indus. Health 271 (1999); W.J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos-A Review.* 39 Indus. Health 57 (2001); R.A.

Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* 10 (2) Int. J. Occupational Envtl. Health (2004); *see also* R. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease.* 45 Am. J. Indus. Med 229 (2004); EPA: *Guidance For Preventing Asbestos Disease Among Auto Mechanics.* (1986); A.H. Smith & C.C. Wright, *Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma.* 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: *Working Safely with Asbestos in Clutch and Brake Linings.* (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. *Asbestos-Automotive Brake and Clutch Repair Work*; World Health Organization, *Environmental Health Criteria 203: Chrysotile Asbestos.* International Programme on Chemical Safety (1998 Geneva)).

Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years with a maximal latency period well over 60 years after the last exposure. Consequently, it could be decades before someone is aware that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover, they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution: In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized

experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer-reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;
b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;
c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's positions vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution*, 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of occupational exposure to asbestos,[1] asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

[1] The remaining 20% of mesothelioma patient likely had asbestos exposures that were para-occupational or are simply unidentified.

<u>Mesothelioma is a dose responsive disease:</u> It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in *Asbestiform Fibers: Non-occupational Health Risks*, published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See Consensus Report, Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution.* 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment.* 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

Malignant mesothelioma is, in general, a dose response disease where each and every significant exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma (*See also* Newman, et al., *Malignant Mesothelioma Register 1987-1999.* 74 Int'l Arch Env. Health 383 (2001), (concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for asbestos disease to develop. Therefore, each non-trivial exposure to asbestos should be considered a substantial contributing factor in the development of the malignant mesothelioma or lung cancer.

<u>Exposure to Asbestos contaminated talc and disease</u>

Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, and the published medical and scientific literature. In 1935 asbestos was identified as a source of exposure in talc miners and millers by Dreesen. By 1968 Cralley had described asbestos in consumer cosmetic talc products. By 1972, the cosmetic industry was looking for asbestos free alternatives to cosmetic talc. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to be contaminated with asbestos. Exposure to asbestos contaminated talc has been shown to cause asbestos related diseases, including mesothelioma. In 1974 Rohl, and in 1976, Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was the same product later tested by Gordon, et.al. Mattenklott et. al. in 2007 found that small amounts of talcum powder (0.1 gram) released thousands of asbestos fibers. A recent paper by Gordon, et.al., Asbestos in Commercial Cosmetic

Talcum Powder as a Cause of Mesothelioma in Women, evaluated the mineralogical constituents of Cashmere Bouquet and its ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper Gordon et.al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions, where anthophyllite and tremolite asbestos were found, regions from which Johnson & Johnson obtained talc for their products. Gordon et.al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et.al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a much lower detection limit with TEM. In addition, the Mine Safety and Health Administration (MSHA) monitored personnel in the mill where Italian talc was ground (this talc was used in consumer products) in 1984. The filters form the personal measurements from these workers contained 5.8% anthophyllite. The MSHA scientist determined that this equated to anthophyllite comprising 0.6% of the bulk Italian talc.

In addition to looking at bulk and air samples, Gordon et.al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to contaminated talcum powder (Cashmere Bouquet). The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring 12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies. Mr. Lanzo, like the woman in this study, had elevated levels of amphibole asbestos, anthophyllite and tremolite, in his lymph node tissue a similar levels.

The issue of asbestos and talc has been studied for decades. Millman 1941 noted pneumoconiosis in a man exposed to cosmetic talc. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung

cancer in miners at the asbestos contaminated talc mines. The International Agency for Research on Cancer has noted that talc contaminated with asbestos is carcinogenic.

Applying an Accepted Method for Evaluating Disease Causation in an Individual
In deciding whether Mr. Lanzo's mesothelioma was caused by his exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health. In this paper, she identifies four questions that should be examined in the causation of disease in an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. With respect to question #1, Mr. Lanzo had an exposure to asbestos from talcum powder for many years, fulfilling this criterion. Johnson and Johnson powders have been shown to contain asbestos and Mr. Lanzo would have had asbestos exposure based on his descriptions from his deposition testimony, his mother's depositon testimony and my interview. Furthermore, asbestos was noted in Mr. Lanzo's lymph nodes, with anthophyllite and tremolite asbestos noted (along with talc). Mr. Lanzo has no known other asbestos exposure. Thus, he has no other competing explanations (#4) for the development of her mesothelioma. The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above, and recently referenced by the Center for Disease Control, there are other individuals with exposure to contaminated talc products who then developed malignant mesothelioma.

Summary and Specific Causation in Mr. Lanzo's Case
Based on the information that was provided to me, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-contaminated cosmetic talc products that Mr. Lanzo used for many decades, starting over 40 years ago, were above normal background levels. Both historic and recent analyses (published in the medical and scientific literature as well as industry, government and private laboratory testing) of the talc from the source mines used by Johnson & Johnson, as well as of Johnson & Johnson products, have shown significant amounts of chrysotile, anthophyllite and tremolite asbestos. Fiber release studies done recently by Fitzgerald and others from products using ore taken from the same source mines as those used in the manufacture of the Johnson & Johnson products showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. Dr. Gordon found anthophyllite and tremolite asbestos at levels of 17,250 fiber/gram wet weight, along with fibrous and platy talc in Mr. Lanzo's lymph

nodes. MSHA found anthophyllite, this same fiber type, in the mills that processed the Italian talc. Similarly, Dr. Compton found anthophyllite in 11 of the 13 samples of talc ore from the Italian mines, from which the talc originated that was then used in consumer products. Mr. Lanzo's exposure to asbestos-contaminated body powder was the cause of his mesothelioma. If he had not used asbestos-containing talcum powder he would not have developed malignant mesothelioma. Alternative powders not containing talc were available since the early 20th century.

The opinions related to Mr. Lanzo's case are based on my review of the evidence of exposure in this case, the medical and scientific literature as described above regarding asbestos exposure and disease, available studies concerning fiber release, epidemiological studies of exposure to asbestos exposure and the development of disease, and my knowledge, skill, experience, and training as a physician specializing in occupational medicine with a clinical focus on evaluating individuals with asbestos exposure.

Mr. Lanzo has metastatic malignant mesothelioma of the pleura. Mr. Lanzo has undergone state of the art treatment, including extensive surgery, that has been shown to increase life expectancy for mesothelioma patients to approximately two years (an increase over chemotherapy alone). However, given that he had metastatic spread of the cancer at the time of surgery, the likelihood of recurrence is high, and carries a decreased prognosis for disease-free survival. In all likelihood, despite his young age and relative good health, his cancer will recur within the next 18-24 months and will, at that point, be fatal. There is no cure for mesothelioma, and despite the aggressive treatment, his prognosis is poor.

I have attached a partial reference list that indicates reliance materials for this report.

Sincerely,

Jacqueline Moline, MD, MSc, FACP, FACOEM

REFERENCE & RELIANCE LIST
Jacqueline Moline, M.D.

1. Andrion, Alberto, et al. *Malignant Peritoneal Mesothelioma in a 17-Year-Old Boy with Evidence of Previous Exposure to Chrysotile and Tremolite Asbestos*, Human Pathology, Volume 25, No. 6 (June 1994).
2. Arul KJ, Holt PF, *Mesothelioma Possibly Due to Environmental Exposure to Asbestos in Childhood*, Int. Arch. Occup. Environ. Hlth 40, 141-143 (1977).
3. *Asbestos, Asbestosis, and Cancer: Helsinki Criteria for Diagnosis and Attribution 2014*, Finnish Institute of Occupational Health (2014).
4. *Asbestos Dust Called a Hazard To at Least One-Fourth of U.S., The New York Times* (March 2, 1966)
5. *Asbestos Fibers Found in Baby Powder*, The Washington Post, March 8, 1976.
6. *Asbestos Found in Ten Powders*, The New York Times, March 10, 1976.
7. *Asbestos: You'll Be All Right if You Just Stop Breathing, New York Times* (July 18, 1971)
8. *Atkinson*, MA., et al. *Evaluation of the Size and Type of Free Particualtes Collected from Unused Asbestos Containing Brake Components as Related to Potential for Respirability*, 46 American Journal of Indusrial Medicine, 545 (2004).
9. Azuma K, Ochiyama I, Chiba Y, Okumura J. *Mesothelioma Risk and Environmental Exposure to Asbestos: Past and Future Trends in Japan.* Int. J. Occup. Environ. Health 15:166-72 (2009).
10. Bamber HA, Butterworth R. *Asbestos hazard from protective clothing*. Ann Occup Hygi 1970, 13:77-79.
11. Barton H, et al., *Assessing Susceptibility from Early-Life Exposure to Carcinogens*, Environmental Health Perspectives 2005; 113 (9); 1125-1133.
12. Bearer C, *How Are Children Different from Adults?*, Environmental Health Perspectives, 103(Suppl 6):7-12 (1995).
13. Birnbaum L, Fenton S, *Cancer and Developmental Exposure to Endocrine Disruptors*, Environmental Health Perspectives 2003; 111 (4); 389-394.
14. Bitchatchi E., et al., *Mesothelioma and asbestosis in a young woman following occupational asbestos exposure: Short latency and long survival: Case Report*, Diagnostic Pathology, 5:81 (2010).
15. Block, L., et al., *Modernization of Asbestos Testing in USP Talc*, U.S. Pharmacopeial Convention, Stimuli to the Revision Process, http://www.usppf.com/pf/pub/data/v404/GEN_STIMULI_404_s201184.html (2014).
16. Blount, A.M., *Amphibole Content of Cosmetic and Pharmaceutical Talcs*, Environmental Health Perspectives, Vol. 94, pp. 225-230 (1991).
17. Boffetta, P., et al. *Cancer Risks Related to Electricity Production*. European Journal of Cancer. 1991; 7 (11); 1504-1519.
18. Boffetta, P., *Epidemiology of Peritoneal Mesothelioma: A Review*, Annals of Oncology 18, 985-990 (2007).
19. Boffetta P. *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment*. Med Lav 1998; 89,6:471-480.
20. British Thoracic Society Standards of Care Committee. *Statement on malignant mesothelioma in the United Kingdom*. Thorax 2001; 56:250-265.
21. Brown, D., et al., *Mortality Patterns Among Miners and Millers Occupationally Exposed to Asbestiform Talc*, Dusts and Diseases, 317-324 (1979).

22. *The Carcinogenicity of Chrysotile Asbestos - A Review,* 39 Industrial Health, 57, 63 (2001).
23. Cherrie JW, Tindall M, Cowie H. *Exposure and risks from wearing asbestos mitts*. Part Fibre Toxicol 2005, 2:5.
24. Churg, *Biological Effects of Asbestos, National Institutes of Health* (2/1/1973)
25. Churg, A. Warnock ML. *Asbestos and other ferruginous bodies.* Am J Pathol 1981; 102:447-56.
26. Coggiola, et al., *An Update of a Mortality Study of Talc Miners and Millers in Italy*, Am. J. Indus. Med. 44:63-69 (2003).
27. Committee on Nonoccupational Health Risks of Asbestiform Fibers, Board on Toxicology and Environmental Health Hazards and Commission on Life Sciences and National Research Council. *Asbestiform Fibers, Nonoccupational Health Risks* (1984)
28. *Consensus Report. Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution.* Scand J Work Environ Health 1997;23:311 – 6.
29. *Consumer Patching Compounds And Artificial Emberizing Materials (Embers and Ash) Containing Respirable Free-Form Asbestos*, Federal Register, Vol. 42, No. 241 (December 13, 1977).
30. Cooke, W.E., *Pulmonary Asbestosis, The British Medical Journal*, pp. 1024-25. (December 3, *1927*)
31. Cralley, J., *Fibrous and Mineral Content of Cosmetic Talcum Products*, Amer. Ind. Hyg. Assoc. J 1968: July-August: 350-354.
32. Cramer DW, Welch WR, et al., *Ovarian Cancer and Talc*, Cancer 50, 372-376 (1982).
33. Dahlgren J, Peckham T. *Mesothelioma Associated With Use of Drywall Joint Compound: A Case Series and Review of Literature*, International Journal of Occupational and Environmental Health, 2012, V.18, No. 4, 337-343.
34. Davis J, et al., *Variations in the Carcinogenicity of Tremolite Dust Samples of Differing Morphology*, The Third Wave of Asbestos Disease: Exposure to Asbestos in Place, The New York Academy of Sciences, Vol 643 (1991).
35. Dement, et al., *Fiber Exposure During Use of Baby Powders, NIOSH* (1972)
36. Dement J, Pompeii L, Lipkus IM, Samsa GP. *Cancer Incidence Among Union Carpenters in New Jersey*, JOEM, Volume 45, Number 10 (October 2003).
37. Dodson, Ronald F., et al, *Asbestos Burden in Cases of Mesothelioma from Individuals from Various Regions of the United States*, Ultrastructural Pathology, 29:5, 415-433 (2005).
38. Dodson, Ronald F., et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review*, 44 American Journal of Industrial Medicine, 291 (2003).
39. Dodson, Ronald F., *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* 39,78-79 (Ronald F. Dodson and Samuel P. Hammar ed), 2006.
40. Dodson, Ronald F. and Hammar, S. <u>Asbestos, Risk Assessment, Epidemiology, and Health Effects, Second Edition</u>. (2011).
41. Doll, *Mortality from Lung Cancer in Asbestos Workers (1955)*
42. Dressen W, Dalla Valle JM, *The Effects of Exposure to Dust in Two Georgia Talc Mills and Mines*, Public Health Reports, Vol. 50, No. 5 (February 1, 1935).
43. Dunning, et al., *Mesothelioma Associated With Commercial Use of Vermiculite Containing Libby Amphibole*, 00 J. Envtl. Med. 00 (March 2012).
44. Egilman D, Fehnel C, Rankin Bohme S. *Exposing the "Myth" of ABC, "Anything But Chrysotile": A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies*. Am Journal Indus Med 44:540-557 (2003).

45. Egilman and Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, Int J Occup Environ Health 2005; 11:360-371.
46. Egilman D. *Fiber Types, Asbestos Potency, and Environmental Causation*. . Int J Occup Environ Health, Vol 15/No. 2, April/June 2009.
47. EPA: Guidance for Preventing Asbestos Disease Among Auto Mechanics. June 1986.
48. EPA: Health Assessment for Talc (March 1992)
49. EPA: National Inventory of Sources and Emissions: Asbestos – 1968. February 1970.
50. Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look,* 52(6) Ann. Occup. Hyg. 455, 460 (2008).
51. Finkelstein, M., *Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State*, Am J Indust Med 55, 863-868 (2012).
52. Finkelstein MM, Meisenkothen C. *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos*. Ann Occ Hyg., Vol. 54, No. 6, pp. 692-696, 2010.
53. Finkelstein, M., *Pneumoconiosis and Malignant Mesothelioma in a Family Operated Metal Casting Business That Used Talc from New York State*, Am J Indust Med 56, 550-555 (2013).
54. Freeman M., Kohles S. *Assessing specific causation of mesothelioma following exposure to chrysotile asbestos-containing brake dust*, International Journal of Occupational and Environmental Health Vol 18, No. 4, 329 (2012).
55. Gamble, et al., *An Epidemiological-Industrial Hygiene Study of Talc Workers,* British Occupational Hygiene Society, 26:1-4 841-85 (1982).
56. Gibbs GW. *Fibre release from asbestos garments*. Ann Occup Hyg 1975, 18: 143-149.
57. Ghio, A, Roggli, V, *Talc Should Not Be Used for Pleurodesis in Patients with Nonmalignant Pleural Effusions,* Am J Respir Crit Care Med, Vol 164, No. 9, pp 1741 (2001).
58. Fujiwara, Hiroshi, et al. *An Autopsy Case of Primary Pericardial Mesothelioma in Arc Cutter Exposed to Asbestos through Talc Pencils*, 43 Industrial Health 346-350 (2005).
59. Gordon, et al., *Asbestos in commercial cosmetic talcum powder as a cause of mesothelioma in women*, 20 International Journal of Occupational and Environmental Health 4, 318-332 (2014).
60. Gordon, *Fertilizer Linked to Worker Death*, Spokesman-Review, June 20, 2001, at 85.
61. Greenberg M. *The Art of Perpetuating a Public Health Hazard*. JOEM Vol 47, No. 2, February 2005.
62. Gunter MR, Belluso E, Mottana A, *Amphiboles: Environmental and Health Concerns*, Reviews in Mineralogy & Geochemistry, Vol. 67, pp. 453-516 (2007).
63. Haque A, et al., *Is There Transplacental Transfer of Asbestos? A Study of 40 Stillborn Infants*, Pediatric Pathology & Laboratory Medicine, 16:877-897, 1996.
64. Hart, et al., *Evaluation of Asbestos Exposures During Firewood-Harvesting Simulations in Libby, MT, USA – Preliminary Data*, Ann. Occup. Hyg., Vol. 51, No. 8, pp. 1-7 (2007).
65. *High Levels of Asbestos Found In 3 Paints and 2 Talcums Here*, New York Times (June 16, 1972).
66. Hillerdal G. *Mesothelioma: new cases associated with non-occupational and low dose exposures.* Occup Environ Med 1999, 56:505-513.
67. Howard, Thomas, *Pneumoconiosis in a Vermiculite End-Product User*, Am. J. Indus. Med. 44:214-217 (2003).
68. Hueper, *Occupational & Non-occupational Exposures to Asbestos, Annals of the New York Academy of Sciences* (1965)
69. Hull, M., el at., *Mesothelioma Among Workers in Asbestiform Fiber-bearing Talc Mines in New York State*, Ann. Occup. Hyg., Vol. 46, Supplement 1, 132-135 (2002).

70. IARC Special Report: *A Review of Human Carcinogens -- Part C: metals, arsenic, dusts, and fibres.* The Lancet Volume 10, May 2009.
71. IARC Monographs on Evaluation of the Carcinogenic Risk of Chemicals to Humans, *Silica and Some Silicates,* Vol. 42 (1987)
72. Ilgren E, et al., *Critical reappraisal of Balangero chrysotile and mesothelioma risk*, Epidemiology Biostatistics and Public Health, Vol. 12, No. 1 (2015).
73. Ilgren E, et al., *Supplemental Data; Critical reappraisal of Balangero chrysotile and mesothelioma risk*, Epidemiology Biostatistics and Public Health, Vol. 12, No. 1 (2015).
74. Imbernon E, Marcharnd JL, Garras L, Goldberg M. *Evaluation quatitative du risqué de cancer du poumon et de mesotheliome pleural chez les mecaniciens de vehicules automobiles. Quantitative assessment of lung cancer and pleural mesothelioma among automobile mechanics*. Rev Epidemiol Sante Publique. 2005, 53:491-500.
75. Iwatsubo, Y., et al., *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-Based Case-Control Study*, 148(2) Am. J. Epidemiology, 133, 136, 141 (1998).
76. Jama, *Asbestosis & Cancer of the Lung* (1949)
77. Joint Policy Committee, Societies of Epidemiology, *Position Statement on Asbestos from the Joint Policy Committee of the Societies of Epidemiology (JPC-SE)*, June 4, 2012.
78. Kanarek MS. *Mesothelioma from Chrysotile Asbestos: Update*. AEP Volume 21, No. 9. September 2011:688-697.
79. Kane M, et al., *Malignant Mesothelioma in Young Adults*, Cancer, Vol. 65(6), 1449-1455, 1990.
80. Karjalainen, Meurman, & Pukkala, *Four Cases of mesothelioma among Finish anthophyllite miners*, Occupational and Environmental Medicine, 51:212-215 (1994).
81. Keyes D.L., et al., *Exposure to Airborne Asbestos Associated with Simulated Cable Installation Above a Suspended Ceiling*, Am. Ind. Hyg. Assoc. J. 52 (November 1991).
82. Kim, Hyoung Ryoul, *Overview of Asbestos Issues in Korea*, 24 J Korean Med Sci 363-7 (2009).
83. Kim, et al., *Lung Cancer Probably Related to Talc Exposure: a Case Report, Industrial Healt*h, 51:228-231 (2013).
84. Kleinfeld, M., et al., *A Study of Workers Exposed to Asbestiform Minerals in Commercial Talc Manufacture*, Environ Research 6, 132-143 (1973).
85. Kleinfeld, M., *Mortality Among Talc Miners and Millers in New York State*, Arch Environ. Health, 14:663-667 (1967).
86. Kleinfeld, M., et al., *Talc Pneumoconiosis*, AMA Archives of Industrial Health, Vol. 12, pp 66-72 (1955).
87. Lacourt A., et al., *Attributable risk in men in two French case-control studies on mesothelioma and asbestos*. Eur. J. Epidemiol (2010) 25:799-806.
88. Lacourt A., et al., *Occupational and non-occupational attributable risk of asbestos exposure for malignant pleural mesothelioma*. Thorax (2014) 0: 1-8.
89. Lamm, S.H., et al., *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers*, Epidemiology-Fibers, 1576-1581, (1988).
90. Landrigan PJ, Nicholson WJ, Suzuki Y, Ladou J. *The Hazards of Chrysotile Asbestos: A Critical Review.* Industrial Health 1999, 37, 271-280.
91. Landrigan PJ, *A Population of Children at Risk of Exposure to Asbestos in Place*, Annals New York Academy of Sciences, 283-286.

92. Landrigan PJ, Collegium Ramazzini, *Comments on the Causation of Malignant Mesothelioma: Rebutting the False Concept That Recent Exposures to Asbestos Do Not Contribute to Causation of Mesothelioma*, Annals of Global Health (2016)
93. Leigh J, Davidson P, Leigh H, Berry D. *Malignant Mesothelioma in Australia, 1945 – 2000*. Am Journal Indus Med, 41:188-201 (2002).
94. Lemen RA. *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model*. Int J Occup Environ Health, Vol 10/No. 2, April/June 2004.
95. Lemen R: *Asbestos in Brakes: Exposure and Risk of Disease*. American Journal of Industrial Medicine, 45:229-237 (2004).
96. Li L, Sun T, Zhang X, Lai R, Li X, Fan X, Morinaga K. *Cohort Studies on Cancer Mortality Among Workers Exposed Only to Chrysotile Asbetsos: a Meta-analysis*. Biomedical and Environmental Sciences 17, 459-468 (2004).
97. Lieben J, et al., *Mesothelioma and Asbestos Exposure*, Arch. Environ. Health, Vol. 14 (April 1967).
98. Lightfoot, Kingston, Pooley, *An Examination of Italian Mine Samples and Relevant Powders, Department of Mineral Exploitation.* (September 8, 1972)
99. Lin S, et al. *Cause-Specific Mortality in Relation to Chrysotile Asbestos Exposure in a Chinese Cohort*. Journal of Thoracic Oncology, Volume XX, Number XX, XXX (2012).
100. Lockey, et al., *Pulmonary Changes After Exposure to Vermiculite Contaminated with Fibrous Tremolite*, Am. Rev. Respir. Dis. (1984).
101. Longo DK, Young RS, *Cosmetic Talc and Ovarian Cancer*, The Lancet, pp 349-351 (Aug 18, 1979).
102. Loomis D, Dement JM, Wolf SH, Richardson DB. *Lung Cancer Mortality and Fiber Exposures among North Carolina Asbestos Textile Workers*. Occup. Environ. Med. published online 11 Mar 2009; doi:10.1136/oem.2008.044362.
103. Lorimer WV, Rohl AN, Miller A, Nicholson WJ, Selikoff IJ. *Asbestos exposure of brake repair workers in the United States*. Mt. Sinai J Med 1976;43:207-18.
104. Lumley KP. *Asbestos dust levels inside firefighting helmets with chrysotile asbestos covers*. Ann Occup Hyg 1971, 14 (3); 285-286.
105. Lynch and Smith, Carcinoma of Lung in Asbestos- Silicosis (1935)
106. Madkour, *Environmental Exposure to Asbestos and the Exposure-Response Relationship with Mesothelioma*, E.Med.Health J. 15:25-38 (2009).
107. Markowitz S, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin Respir Crit Care Med, 36:334-346 (2015).
108. Mattenklott, M., TRANSLATION OF: “Asbestos in talc powders and soapstone – the present state” --- Asbest in Talkumpudern und Speckstein – heutige Situation. Gefahrstoffe – Reinhalt. Luft 67 (2007) no. 7/8 p. 287-291. (by courtesy of Springer-VDI-Verlag, Düsseldorf).
109. Mazurek, et al., Malignant Mesothelioma Mortality – United States, 1999-2015, Centers for Disease Control and Prevention: Morbidity and Mortality Weekly Report, 66(8);214-218 (March 3, 2017).
110. McDonald, et al., Malignant Mesothelioma in North America, 46 Cancer 7 (Oct. 1, 1980).
111. McDonald, et al., Mesothelioma and Asbestos Fiber Type: Evidence from Lung Tissue Analysis, Cancer, Vol. 63, No. 8, 1544-1547 (1989).
112. Merewether & Price, Report on Effects of Asbestos Dust on Lung & Dust Suppression in Asbestos Industry (1930)
113. Mesothelioma in a Brake Repair Worker, The Lancet. November 13, 1982.

114. Meurman, Kiviluoto, & Hakama, *Mortality and morbidity among the working population of anthophyllite asbestos miners in Finland*, British Journal of Industrial Medicine, 31:105-112 (1974).
115. Meurman, Pukkala, Hakama, *Incidence of Cancer among Anthophyllite asbestos miners in Finland*, Occupational and Environmental Medicine, 51:421-425 (1994).
116. Miller R., How Environmental Hazards in Childhood Have Been Discovered: Carcinogens, Teratogens, Neurotoxicants, and Others, Pediatrics 2004;113;945.
117. Millman N, Pneumonoconiosis Due To Talc In The Cosmetic Industry, Occup. Med., 4:391 (1947).
118. Mirabelli D, Cavone D, Merler E, Gennaro V, Romanelli A, Mensi C, Chellini E, Nicita C, Marinaccio A, Magnani C, Musti M. Non-occupational exposure to asbestos and malignant mesothelioma in the Italian National Registry of Mesotheliomas. Occup Environ Med 2010;67:792e794. doi:10.1136/oem.2009.047019.
119. Mirabelli D, Cadum E, Mortality among patients with pleural and peritoneal tumors in Alta Valle di Susa, Epidemiol Prev., 26(6):284-6 (Nov-Dec 2002).
120. Newhouse and Thompson. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 1965, 22 (4): 261-269.
121. New York Industrial Code Rule No. 12, adopted TLV. 1958
122. Nicholson WJ, Rohl A, Fischbein A, Selikoff IJ. Occupational and Community Asbestos Exposure from Wallboard Finishing Compounds. Bull. N.Y. Acad. Med., Vol. 51, No. 10, November 1975.
123. Nicholson WJ, Landrigan PJ. Asbestos: A Status Report. Current Issues in Public Health 1996, 2:118-123.
124. Nicholson WJ. The Carcinogenicity of Chrysotile Asbestos – A Review. Industial Health 2001, 39, 57-64.
125. Nynas, et al., *Cancer Incidence in Asbestos-Exposed Workers: An Update On Four Finnish Cohorts*, Finnish Institute of Occupational Health, 8:169-174 (2017).
126. Offermans N., et al., Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study, J. Occ. Environ. Medicine, Vol. 56:1 (Jan. 2014).
127. Olsen, NJ, et al. Increasing Incidence of Malignant Mesothelioma After Exposure to Asbestos During Home Maintenance and Renovation. Med J Aust 2011; 195 (5): pp. 271-274.
128. Oury, T., Sporn, T., and Roggli, V, <u>Pathology of Asbestos-Associated Diseases,</u> Third Ed. Copyright 2014 – Chapters 3 and 8.
129. Paoletti, L., Evaluation by Electron Microscopy Techniques of Asbestos Contamination in Industrial, Cosmetic and Pharmaceutical Talcs, Regulatory Toxicology and Pharmacology, 4:222-235 (1984).
130. Pearce N, Checkoway H, Kriebel D: Bias in Occupational Epidemiology Studies, Occup Environ Med 2007;64:562-568. doi: 10.1136/aem.2006.026690.
131. Peretti, Luigi, Bulletin of the Subalpine Mining Association - Year III No. 3-4, "*Geology and Genesis of the Talc Deposits in the Pinerolese,*" September - December 1966.
132. Phillips, Murray, *Malignant Mesothelioma in a Patient with Anthophyllite Asbestos Fibres in the Lungs*, Oxford University Press, 54:412-416 (2010).
133. Piolatto, Giorgio, et al. An Update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy, 47 British Journal of Industrial Medicine 810-814 (1990).
134. Pira, et al., Mortality of Talc Miners and Millers from Val Chisone, Northern Italy, An Updated Cohort Study, Journal of Occupational and Environmental Medicine, Volume 59, Number 7 (July 2017).

135. Pooley, F., et al., Chemical and Physical Properties of British Talc Powders, Inhaled Particles, 639-646 (1975).
136. Porro FW, et al., Pneumoconiosis in the Talc Industry, Am. J. Roentgenology and Radium Therapy, Vol. 47, No. 4 (April 1942).
137. Price, B., Industrial-Grade Talc Exposure and the Risk of Mesothelioma, Crit Rev Toxicol 40(6), 513-530 (July 2010).
138. Ramazzini, Collegium, Asbestos is Still With Us: Repeat Call for A Universal Ban, 16 Int. J. Occup. Environ. Health, 351, 351-352 (2010).
139. Reid A, et al., Mesothelioma risk after 40 years since first exposure to asbestos: a pooled analysis, Thorax (2014).
140. Rodelsperger K, Jockel KH, Pohlabein H, Romer W, Woitowitz HJ. Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results From a German Hospital-Based Case-Control Study. Am Journal of Ind Med, 39:262-275 (2001).
141. Roelofs, Cora, et al., Mesothelioma and employment in Massachusetts: Analysis of cancer registry data 1988 – 2003, American Journal of Industrial Medicine (2013)
142. Roggli, Victor, *Pathology of Human Asbestosis: A Critical Review*, Adv Pathol 2:31-60 (1989).
143. Roggli, Victor, et al., *Pulmonary Asbestos Body Counts and Electron Probe Analysis of Asbestos Body Cores in Patients with Mesothelioma, A Study of 25 Cases,* Cancer 50:2423-2432 (1982).
144. Roggli, Victor, et al., Tremolite and Mesothelioma, Ann. Occup. Hyg., Vol. 46, No. 5, pp. 447-453 (2002).
145. Rohl, AN, Asbestos in Talc, Enviro Health Persp, Vol 9, pp 129-132 (1974).
146. Rohl AN, Langer AM, Selikoff IJ, Nicholson WJ. Exposure to Asbestos in the Use of Consumer Spackling, Patching, and Taping Compounds. Science, 15 August 1975, Volume 189, pp. 551-553.
147. Rohl AN, Langer A, Fibrous Mineral Content of Consumer Talc-Containing Products, pp 394-403 (1979).
148. Rohl AN, Langer A, Identification and Quantitation of Asbestos in Talc, Enviro Health Persp, Vol 9, pp 95-109 (1974).
149. Rohl, A., et al. Asbestos Content of Dust Encountered in Brake Maintenance and Repair, 70 Proceedings of the Royal Society of Medicine, 32(1977).
150. Rohl, A., Langer, A., et al., Consumer Talcums and Powders: Mineral and Chemical Characterization, Journal of Toxicology and Environmental Health, 2:255-284 (1976).
151. Rohs, et al., Low-Level Fiber-induced Radiographic Changes Caused by Libby Vermiculite, A 25-Year Follow-up Study, 177 Am. J. Respir. Crit. Care. Med. 630 (2008).
152. Rolland, P., et al, "Risk of Pleural Mesothelioma: A French Population-Based Case-Control Study (1998-2006)." October 20, 2006/ Oral Session: Epidemiology II.
153. Rom, et al., *Malignant Mesothelioma From Neighborhood Exposure to Anthophyllite Asbesto*s, American Journal Of Industrial Medicine, 40:211-214 (2001).
154. Rubino, G.F., et al. Mortality Study of Talc Miners and Millers, Department of Occupational Medicine University of Torino Italy, presented at American Industrial Health Conference, San Francisco, California (April 14, 1975).
155. Rubino, G.F., et al. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy, 36 British Journal of Industrial Medicine 187-194 (1979).
156. Saitoh, Muto, Hachiya, Takizawa, Asbestos Body and Fiber Concentrations in Pathological Autopsy Tissues of Patients with Malignant Peritoneal Mesothelioma, Bull. Environ. Contam. Toxicol. 50:325-332 (1993).

157. Samimi BS, Williams AM. Occupational exposure to asbestos fibres resulting from use of asbestos gloves. Am Ind Hyg Assoc J 1981, 42:870-875.
158. Scancarello G, Romero R, Respiratory Disease as a Result of Talc Inhalation, J Occup Environ Med 38(6), 610-614 (June 1996).
159. Schepers, GW H and Durkan, TM, An Experimental Study of the Effects of Talc Dust on Animal Tissue, American Medical Association's Archives of Industrial Health, Vol. 12, Number 6. (December 1955)
160. Schulz RZ, Williams CR, Commercial Talc Animal and Mineralogical Studies, J Ind Hygiene and Toxicology, Vol. 24, No. 4 (1942).
161. Sebastien, Janson, Gaudichet, Hirsch and Bignon, Asbestos retention in Human Respiratory Tissues: Comparative Measurements in Lung Parenchyma and in Parietal Pleura, 30 IARC Sci. Pub., 237-246 (1980).
162. Silvertstein, M., Welch, L., Lemen, R. Developments in Asbestos Cancer Risk Assessment. Am Journal of Ind Med, 2009.
163. Smith AH, Wright CC. Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma. Am J Indus Med 30:252-266 (1996).
164. Smith, D., Women and Mesothelioma, Chest, 122/6 1885-1886 (2002).
165. Srebo S, Roggli, V, Asbestos-Related Disease Associated with Exposure to Asbestiform Tremolite, Am J Int Med 26, 809-819 (1994).
166. Stayner LT. Canada's Asbestos International Expert Panel Report. J Occup Environ Med, 22.12.2008.
167. Suzuki Y, Kohyama N. Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues. Am J Indus Med., 19:701-704 (1990).
168. Suzuki, Y. & Kohyama, N. Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues, 19 Am. J. Ind. Med, 701 (1991).
169. Suzuki Y, Yuen SR. Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma. Annals of the New York Academy of Science, Volume 982, December 2002.
170. Suzuki Y, Yuen SR, Ashley R. Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence. Int J Hyg Environ-Health 208 (2005) 201-210.
171. Teschke, et al., Mesothelioma Surveillance to Locate Sources of Exposure to Asbestos, Can. J. Pub. Health (May – June 1997).
172. Thomas, *Lung Cancer Mortality among pottery workers in the United States*, Lyon International Agency for Research on Cancer (1990).
173. Thomas & Stewart, *Mortality From Lung Cancer and Respiratory Among Pottery Workers Exposed To Silica*, American Journal of Epidemiology, (1987).
174. Transactions – National Safety Council (NSC 3) (1934).
175. US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, NIOSH. Control of Asbestos Exposure During Brake Drum Service. August 1989.
176. US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, NIOSH. Report to Congress on Workers' Home Contamination Study Conducted Under the Workers' Family Protection Act (29 U.S.C. 671a). September 1995.
177. US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, NIOSH. Asbestos Fibers and Other Elongate Mineral Particles: State of the Science and Roadmap for Research Revised Edition, 1-174 (2011).
178. U.S. Dept. of Labor: Working Safely with Asbestos in Clutch and Brake Linings. (posting).

179. U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. Asbestos – Automotive Brake and Clutch Repair Work (bulletin).
180. U.S. Environmental Protection Agency – Region IX, *Response to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group, Inc. "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project,"* April 20, 2006.
181. Van Gosen, Bradley, The Geology of Asbestos in the United States and Its Practical Applications, Environmental & Engineering Geoscience, Vol. XIII, No. 1, February 2007. pp. 55-63.
182. Van Gosen, Bradley, et al., Using the geologic setting of talc deposits as an indicator of amphibole asbestos content, 45 Environmental Geology 920-939 (2004).
183. Van Horn, Earl C. *Talc Deposits of the Murphy Marble Belt*," NC Dept of Conservation and Development, Bulletin Number 56 (1948).
184. Vianna NJ. Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Females. The Lancet, May 20, 1978.
185. Vinikoor, et al., Exposure to Asbestos-Containing Vermiculite Ore and Respiratory Symptoms Among Individuals Who Were Children While the Mine Was Active in Libby, Montana, 118 Envtl. Health Perspectives 7 (July 2010).
186. Wagner, Diffuse Pleural Mesothelioma & Asbestos Exposure in the Northwestern Cape Province (1960)
187. Wagner, J.C., et al., Biological Effects of Tremolite, 45 Br. J. Cancer 352 (1982).
188. Ward, et al., Trees as reservoirs for amphibole fibers in Libby, Montana, Science of the Total Environment 367, pp. 460-465 (2006).
189. Webber, et al., Separation and Characterization of Respirable Amphibole Fibers from Libby, Montana, Inhalation Toxicology, 20:733-740 (2008).
190. Weill, H, et al., Health Effects of Tremolite, Am Rev Respir Dis 142, 1453-1458 (1990).
191. Welch, Laura, et al. Asbestos and Peritoneal Mesothelioma among College-educated Men, 11 International Journal of Occupational Environmental Health 254-258 (2005).
192. Welch, et al, Asbestos Exposure Causes Mesothelioma, But Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, Int J Occup Environ Health 2007; 13:318-327.
193. Whitehouse, et al., Environmental Exposure to Libby Asbestos and Mesotheliomas, Am. J. Indus. Med. 50:877-880 (2008).
194. Wild C, Kleinjans J, Children and Increased Susceptibility to Environmental Carcinogens: Evidence or Empathy?, Cancer Epidemiology, Biomarkers & Prevention, 2003, Vol. 12; 1389-1394.
195. World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. International Programme on Chemical Safety 1998. Geneva.
196. World Health Organization. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 93, Carbon Black, Titanium Dioxide, and Talc, Lyon, France (2010).
197. Yano, Wang, Wang, Wang, Lan, Cancer Mortality Among Workers Exposed to Amphibole-Free Chrysotile Asbestos, American J. Epid. 15, 154(6):538-43 (Sept. 2001).
198. Yano E, et al., Mesothelioma in a Worker Who Spun Chrysotile Asbestos at Home During Childhood, American Journal of Industrial Medicine, 52:282 – 287 (2009).

SELECTION OF SPECIFIC MATERIALS REVIEWED/RELIED ON:

A. Report of Dr. Steven Compton, MVA Scientific Consultants, August 1, 2017
B. Report of Dr. William Longo and Dr. Mark Rigler, Materials Analytical Services, LLC, August 2, 2017

C. Cosmetic Toiletry & Fragrance Association documents
D. Product and/or Source Talc Testing produced in litigation (see list below)

1. October 15, 1957 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000001032-JNJAZ55_000001065.
2. March 23, 1958 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000001341-JNJNL61_000001368.
3. May 9, 1958 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000010804-JNJNL61_000010843.
4. March 5, 1959 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000001114-JNJAZ55_000001120.
5. July 31, 1959 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000000840-JNJAZ55_000000904.
6. August 31, 1959 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000009799-JNJNL61_000009813.
7. December 31, 1959 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000000797-JNJAZ55_000000839.
8. January 31, 1960 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000002190-JNJAZ55_000002202.
9. March 8, 1960 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000002203-JNJAZ55_000002219.
10. April 12, 1960 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000001480-JNJNL61_000001484.
11. July 11, 1961 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000001464-JNJNL61_000001471.
12. October 13, 1961 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000001014-JNJAZ55_000001018.
13. July 13, 1966 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000000049-JNJAZ55_000000051.
14. January 16, 1967 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000009897.
15. January 30, 1967 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJNL61_000009896.

16. April 17, 1967 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000001725-JNJNL61_000001726.
17. January 24, 1968 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000001489-JNJNL61_000001493.
18. October 31, 1968 – Johns-Manville Corporation testing, Body Talcum Powders – Petrographic Examination
19. June 30, 1971 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJAZ55_000006060.
20. July 7, 1971 – Colorado School of Mines Research Institute Report on Vermont Talc to Johnson & Johnson
21. July 19, 1971 – FDC Report on Bureau of Foods finding asbestos in Johnson & Johnson
22. July 29, 1971 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJMX68_000004646-JNJMX68_000004647.
23. February 1972 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJNL61_000001785.
24. May 15, 1972 – International Technical Service Manufacturing Letter for Body Powder
25. June 12, 1972 – ES Lab test of 1615 talc. WCD 1615 Talc Analysis (ASBESTOS)
26. June 12, 1972 – ES Lab test of Montana talc. WCD Talc Analysis (ASBESTOS)
27. July 14, 1972 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
28. August 3, 1972 – Letter and final analytical results from S. Lewin to A. Weissler
29. August 24, 1972 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000003576-JNJAZ55_000003577.
30. September 30, 1972 – Lewin test of 1615 talc. WCD 1615 Talc Analysis (ASBESTOS)
31. October 27, 1972 – McCrone Examination of Johnson and Johnson's Baby Powder
32. October 27, 1972 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000005977-JNJAZ55_000005979.
33. December 8, 1972 – Pfizer test of 1615 talc. Letter to Clements from Stanley, Asbestos in Talc- findings from diffraction
34. April 27, 1973 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJAZ55_000017891.
35. May 8, 1973 – Proceedings of the Symposium on Talc, Washington DC by A. Langer
36. May 16, 1973 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000001892-JNJAZ55_000001898.
37. June 4, 1973 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJAZ55_000005083; JNJAZ55_000001587.

38. June 6, 1973 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJAZ55_000004643.
39. June 6, 1973 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Number JNJMX68_000006483.
40. July 31, 1973 – Memorandum from A. Weissler to R. Shaffner
41. August 28, 1973 – JM's TEM talc testing/ 14 samples. Memo to R.S. Lamar from V.E. Wolkodoff, Subject: "Determination of Chrysotile by TEM in Commercial Talcs."
42. October 19, 1973 – WCD Bristol-Myers Products Letter - Six samples of talc
43. December 10, 1973 – Round Robin test of Italian, Montana, Alabama, Vermont, and North Carolina talc. "Report of CTFA Method to Detect Chrysotile and Tremolite in Talc"
44. May 14, 1974 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJMX68_000004996-JNJMX68_000005044.
45. May 1, 1975 – RT Vanderbilt Company, Material Safety Data Sheet
46. 1976 - Briscese Lab Notebook. Colgate testing of Italian, Montana, North Carolina talc
47. March 1, 1976 - Dr. Shapiro and Dr. Langer "Memorandum for the Record of Telephone Conversation."
48. March 5, 1976 – Pfizer testing of two talc samples, Airspun fact powder [Code D070] and Airspun face powder [Code C-213].
49. December 1976 through June 1978 – Pfizer memos re testing talc samples to Coty Divisions of 1615 and 141 talcs
50. 1977 - McCrone Test of 1615 talc. Dates: 1970s-1980s
51. June 13, 1977 Minutes of CTFA Task Force on Round Robin Testing of Consumer Talcum Products for Asbestiform Amphibole Minerals
52. October 13, 1977 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
53. January 24, 1978 – McCrone test of Vermont talc. WCD Talc Analysis (ASBESTOS)
54. May 26, 1978 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
55. August 25, 1978 – McCrone test of Vermont talc. WCD Talc Analysis (ASBESTOS)
56. November 14, 1978 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
57. February 13, 1979 – McCrone test of Vermont talc. WCD Talc Analysis (ASBESTOS)
58. March 5, 1979 – McCrone test of Vermont talc. WCD Talc Analysis (ASBESTOS)
59. November 1, 1979 – Pfizer memo re. "Routine Analysis of Coty Talc for Fibrous Tremolite" in 1615 and 141 talc samples
60. January 12, 1980 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000009483-JNJNL61_000009485.
61. September 17, 1980 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
62. March 16, 1981 – McCrone Test of Monoblend for Colgate-Palmolive Co.
63. May 15, 1984 – Report of MSHA visit to Cyprus Industrial Minerals Company South Plainfield Mill
64. July 7, 1987 – ES Lab test of Vermont talc. WCD Talc Analysis (ASBESTOS)
65. October 19, 1988 – Due Diligence of for Windsor Minerals, Inc.
66. February 24, 2004 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJI4T5_000004096-JNJI4T5_000004100.

67. July 9 – Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJAZ55_000005743-JNJAZ55_000005748.
68. Key to testing samples from Blount, A.M. (1991) Bates Number JNJNL61_000014437
69. Key to testing samples from Blount, A.M. (1991) Bates Number GUNTER00002233
70. Protected Document, *Irma Verdolotti v. Brenntag North America, Inc., et al.*, Superior Court of New Jersey for Middlesex County, Docket No.: MID-L-5973-16, Bates Numbers JNJNL61_000005343.